atric squeal developed at the one patient.

Conclusion: The cases in the forefront of change in mental status viral meningoencephalitis should be considered and empirical treatment with acyclovir should be started.

http://dx.doi.org/10.1016/j.ijid.2012.05.202

Type: Poster Presentation

Final Abstract Number: 40.041
Session: Virology and Viral Infections (Non-HIV)
Date: Thursday, June 14, 2012
Time: 12:45-14:15
Room: Poster & Exhibition Area

Is lymphoepithelioma like carcinoma of thymic caused by ebv infection?

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Background: Association between EBV infection and occurrence of certain cancer including lymphomas, carcinoma of salivary gland, lung, nasopharynx and gastric is well documented. The causative role of EBV in these cancer is uncertain and is the matter of debate. However monoclonal proliferation of EBV-positive tumour cells in some cancers strongly suggest that EBV infection proceeded neoplastic transformation and may play some role in the early stage of carcinogenesis.

Methods: In this report we described the case of lymphoepithelioma like carcinoma of thymic with the presence of EBV.

Results: 17-years old boy has been admitted to hospital due to fever over 390C, enlargement of cervical lymph nodes on the left side. CT and MRI examination showed in mediastinum big tumour mass consisting of the enlarged thymus and conglomerate of lymph nodes. Among the additional tests the following abnormalities were found: elevated levels of LDH and presence of IgG and IgM antibodies to EBV. Histological examination of cervical lymph node revealed metastases of lymphoepithelioma like carcinoma of thymic. Quantitative real-time with labeled probe and primers specific for EBV showed high copy number of EBV in tumour cells as well as in the serum. Interestingly no EBV has been detected in peripheral blood lymphocytes. Pretreatment copy number of EBV was so high as 4.56x108 copies/ml in serum and 2.1x107 copies/1μg of carcinoma cells. Subsequent quantitative EBV measurement in the serum has been performed before each chemotherapy course. The level of EBV in the serum gradually decreased reaching 2.4x106 before second course and 3.7x103 before the third course. Finally EBV disappeared completely from the serum before fourth course of chemotherapy. MRI examination after third chemotherapy course showed only thymus enlargement without pathologic lymph nodes.

Conclusion: The presence of EBV in tumor cells and absence in peripheral blood lymphocytes indicated that serum EBV particles originates from carcinoma cells. Therefore we can concluded, that it is very likely, that EBV contributes to thymic lymphoepithelioma like carcinoma development. In addition we can suggest that EBV presence in the serum can be additional cancer marker, indicating on effectiveness of chemotherapy.

http://dx.doi.org/10.1016/j.ijid.2012.05.203

Final Abstract Number: 40.042
Session: Virology and Viral Infections (Non-HIV)
Date: Thursday, June 14, 2012
Time: 12:45-14:15
Room: Poster & Exhibition Area

Furious and paralytic rabies: glyco-/nucleo-/phosphoprotien nucleotide sequence heterogeneities at different parts of central nervous system

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Background: Previous studies showed that furious rabies has greater viral burden in brain with less pronounced immune response than in the paralytic form. Interruption of axonal tracts by inflammation has been found at brainstem of paralytic dogs. Presence of quasispecies has not been found as determined by sequencing of viral isolates from one central nervous system (CNS) region.

Methods: The nucleo- (N), phospho- (P) and glyco-protein (G) genes of rabies viruses (RV) from 4 different CNS regions [frontal lobe, hippocampus, pons, spinal cord (SC)] were characterized. Samples were obtained from 3 paralytic and 3 furious dogs while animals were still conscious.

Results: Sequence heterogeneities were found at a greater degree in paralytic than furious dogs when viruses isolated from SC were compared with those from brainstem and cerebrum. The average number of nucleotide (nt) and amino acid (aa) differences of the G gene ranged from 13.7 nt (2.7 aa) in furious to 23 (4.7) in paralytic between SC and pons; 15 (2.7) to 24 (5) between SC and hippocampus; and 19.3 (5.3) to 27.7 (8) between SC and frontal lobe. Mutations were mostly in the signal transduction region and nAchR binding domain, but not in PDZ-BS which plays a role in apoptosis. Viruses of furious and paralytic dogs carried pathogenic determinants in G gene. Similarly, differences in N gene were 7 nt (4 aa) to 12 nt(4 aa), 11 (6.3) to 8 (2), and 7 (3.7) to 9.7 (4.7). Mutations in P genes were found exclusively in paralytic rabies; 5.3 nt (2 aa) between SC and pons, 10.3 (1.7) between SC and hippocampus, and 8.3 (1.3) between SC and frontal lobe. Most occurred at dynein light chain binding sites (DYNLL1 and DYNLL2) for virus transport.

Conclusion: It appears that as viruses ascend from SC to brain, genetic as well as host responses take place. This needs further study.

http://dx.doi.org/10.1016/j.ijid.2012.05.204